GINGIVAL & PERIODONTAL DISEASES IN CHILDREN
INTRODUCTION

* The **periodontium** (peri-around, dontium-tooth, in Greek) consists of investing and supporting tissues.
* Investing tissue of the periodontium- **gingiva**.
* Tooth and periodontium together- **dentoperiodontal** unit.
* Tissue includes gingiva, PDL, cementum, alveolar process.
* Shorter life span of primary teeth is one of the reasons why little attention is paid to periodontitis in children.

* Rate of periodontal destruction in children is much slower to cause substantial loss of attachment within the life span of primary teeth.
HISTORY

* Ancient Chinese medical works discussed.
* McCall (1938) said gingivitis can be observed in children as young as 4-5 years old.
* Page & Schroeder (1982)- 4 forms: adult periodontitis (AP), rapidly progressive periodontitis (RPP), juvenile periodontitis (JP), and prepubertal periodontitis (PP).
* Watanabe (1990)- prepubertal periodontitis was caused by Prevotella intermedia, Porphyromonas gingivalis, Capnocytophaga and Eikenella corrodens.
GINGIVA

* Gingiva is the part of oral mucosa that covers the alveolar processes of jaws and surrounds the neck of teeth.

(CARRANZA)
Marginal / Unattached Gingiva in Adults-
1. Border surrounds in collar like fashion.
2. Demarcated from adjacent attached gingiva by free gingival groove. 1mm width. Knife-edged.
3. Forms soft tissue wall of sulcus.

Attached Gingiva in Adults-
1. Firm and resilient and tightly bound to the underlying periosteum.
2. Width- Maxillary incisor- 3.5 to 4.5mm, Mandible incisor- 3.3 to 3.9mm.
3. Least width in first premolar region (1.8-1.9 mm)
4. Width of attached gingiva increases with age
PRIMARY DENTITION-

1. Marginal gingiva is thicker & rounder due to morphological characteristics such as *cervical bulge* & underlying constriction at CEJ.

2. Less dense & redder- less keratinized epithelium & greater vascularity.

3. More flaccid & less stippled.

4. Two unique characteristics – interdental clefts & retropuspid papilla
PRIMARY DENTITION-

* **Retrocuspid papilla**- on attached gingiva lingual to mandibular canine, usually bilateral. Occurs in 85% children

* Width of attached gingiva is greater in incisor area, decreases over cuspids, increases again over primary & permanent molars.

* Interdental spacing is common resulting in **saddle areas**, well keratinized interdental surface reason for lower prevalence of periodontal lesions in children.

* In primary teeth sulcus depth is greater than that found around permanent teeth. Mean values - 1.4mm to 2.1mm.
<table>
<thead>
<tr>
<th>Gingiva in children</th>
<th>Gingiva in adults</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marginal gingiva</td>
<td>Marginal gingiva</td>
</tr>
<tr>
<td>round, thick, rolled, flaccid, retractable.</td>
<td>Knife edge margin seen</td>
</tr>
<tr>
<td>Attached gingiva</td>
<td>Attached gingiva</td>
</tr>
<tr>
<td>wider, less stippling. Less dense, redder, 2 unique features- interdental clefts, retrocuspid papillae.</td>
<td>More stippled, coral pink.</td>
</tr>
<tr>
<td>Papillary gingiva</td>
<td>Papillary gingiva</td>
</tr>
<tr>
<td>Gingival Sulcus</td>
<td>Gingival Sulcus</td>
</tr>
<tr>
<td>more (1.4-2.1 mm)</td>
<td>Less.</td>
</tr>
</tbody>
</table>
Characteristics of Normal Periodontium in Children

COLOUR-
* Pink or red.
* Blacks- deep purplish discoloration seen.

CONSISTENCY-
* Attached gingiva- flaccid & retractable.

CONTOUR-
* Collarlike fashion and a scalloped outline

SHAPE-
* Anteriorly- pyramidal, posteriorly-flattened
**SURFACE TEXTURE**

- Absent in infancy, 2-3 years of age, increases until adulthood, disappears in old age.
- Microscopically, stippling is produced by alternate rounded protuberances & depressions in the gingival surface.

- **Anatomic crown** (portion of tooth covered by enamel) & **anatomic root** (portion of tooth covered by cementum).
- **Clinical crown** (part of tooth denuded of its gingiva) & **clinical root** (portion of tooth which is covered by periodontal tissue).
PHYSIOLOGIC GINGIVAL CHANGES ASSOCIATED WITH TOOTH ERUPTION

* Pre-eruption bulge: Before crown appears, gingiva presents firm bulge, slightly blanched and confirms to contour of underlying crown.
* Formation of gingival margin: Marginal gingiva and sulcus develop as crown penetrates oral mucosa.
* In course of eruption, gingival margin is edematous, rounded, and slightly reddened.

GINGIVITIS

- Gingivitis is inflammation of the gingiva that does not result in clinical attachment loss. It is a reversible disease.
- Dental plaque induced gingival inflammation is the most common form of gingivitis.
- Although the microbiology of this disease has not been completely characterized, increased subgingival levels of Actinomyces sp., Capnocytophaga sp., Leptotrichia sp., and Selenomonas sp. have been found in experimental gingivitis in children when compared to gingivitis in adults.

Gingivitis associated with poor oral hygiene is usually classified into four stages:

* Stage 1: Initial lesion
* Stage 2: Early lesion
* Stage 3: Moderate lesion
* Stage 4: Advanced lesion
<table>
<thead>
<tr>
<th>Stages</th>
<th>Days</th>
<th>Vascular changes</th>
<th>Predominant immune cells</th>
<th>Clinical findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage I</td>
<td>2-4</td>
<td>↑ permeability of vascular bed</td>
<td>PMNs</td>
<td>↑ Gingival fluid flow</td>
</tr>
<tr>
<td>Stage II</td>
<td>4-7</td>
<td>Vascular proliferation</td>
<td>lymphocytes</td>
<td>Erythema, Bleeding on probing</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage III</td>
<td>14-21</td>
<td>Stage II + Blood stasis</td>
<td>Plasma cells &amp; B lymphocyte</td>
<td>Change in colour, size, texture, etc.</td>
</tr>
<tr>
<td>Stage IV</td>
<td>&gt;month</td>
<td>Degeneration</td>
<td>Plasma cell</td>
<td>Loss of connective tissue attachment and alveolar bone</td>
</tr>
</tbody>
</table>
CLASSIFICATION OF GINGIVAL AND PERIODONTAL DISEASES
(International workshop for classification of Periodontal Diseases & Conditions 1999)

I. Gingival Diseases
   - A. Dental plaque-induced gingival diseases
   - B. Non-plaque-induced gingival lesions

II. Chronic Periodontitis (slight: 1-2 mm CAL; moderate: 3-4 mm CAL; severe: > 5 mm CAL)
   - A. Localized
   - B. Generalized (> 30% of sites are involved)

III. Aggressive Periodontitis (slight: 1-2 mm CAL; moderate: 3-4 mm CAL; severe: > 5 mm CAL)
   - A. Localized
   - B. Generalized (> 30% of sites are involved)
IV. Periodontitis as a Manifestation of Systemic Diseases

* A. Associated with hematological disorders
* B. Associated with genetic disorders
* C. Not otherwise specified

V. Necrotizing Periodontal Diseases

* A. Necrotizing ulcerative gingivitis
* B. Necrotizing ulcerative periodontitis

VI. Abscesses of the Periodontium

* A. Gingival abscess
* B. Periodontal abscess
* C. Pericoronal abscess
VII. Periodontitis Associated With Endodontic Lesions

A. Combined periodontic-endodontic lesions

VIII. Developmental or Acquired Deformities and Conditions

A. Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis

B. Mucogingival deformities and conditions around teeth

C. Mucogingival deformities and conditions on edentulous ridges

D. Occlusal trauma
A. Gingival Diseases Associated With Plaque

I. Without Local Contributing Factor

* Plaque - Induced Gingivitis
* The primary cause of gingivitis is plaque.
* Dental plaque appears to form more rapidly in children aged 8 to 12 years than in adults.
**Clinical Features**

* The plaque-induced inflammatory lesion is usually confined to the marginal aspects of the gingiva.
* A fiery red surface discoloration is often seen.
* Gingival color change and swelling appear to be more common expressions of gingivitis in children than are bleeding and increased pocket depth.
* Long term exposure can cause plaque induced gingival enlargement also.
Therapy for individuals with chronic gingivitis is initially directed at reduction of oral bacteria and associated calcified and noncalcified deposits.

Many patients with gingivitis have calculus or other associated local factors.

Removal of dental calculus is accomplished by scaling and root planning procedures including reducing subgingival calculus.

Use of topical antibacterial agents is recommended.

TREATMENT

- Three medicaments have been given the ADA Seal of Acceptance for the control of gingivitis.
- The active ingredients of one product are thymol, menthol, eucalyptol, and methyl salicylate.
- Active ingredients in the other two are chlorhexidine digluconate and triclosan.
- These agents are useful for the control of supragingival, but not subgingival plaque.
- The success is determined by evaluating the periodontal tissues following treatment and during the maintenance phase of therapy.
With Local Contributing Factors

* Eruption Cyst & Hematoma

* It is common for erupting teeth to be associated with a form of dentigerous cyst called an eruption cyst.

* It is usually translucent, fluctuant and circumscribed swelling.

* When cystic cavity contains blood, swelling appears as purple/deep-blue fluctuant, circumscribed swelling termed as eruption hematoma.
Gingivitis associated with tooth eruption is frequent.

Tooth eruption does not cause gingivitis. But, due to plaque accumulation in areas of shedding primary teeth and erupting permanent teeth.

The inflammatory changes accentuate the normal prominence of the gingival margin and create the impression of a marked gingival enlargement.
Gingivitis Associated With Orthodontic Appliance

* Access of interproximal tooth brushing is reduced due to fixed appliance therapy.
* Usually banded rather than bonded.
* Supragingival plaque deposits are shifted into a subgingival location by tipping movement.
* Thus, gingival changes can occur within 1-2 months of appliance placement and are generally transient.
B. GINGIVAL DISEASES MODIFIED BY SYSTEMIC FACTORS

I. ASSOCIATED WITH ENDOCRINE SYSTEM

PUBERTY GINGIVITIS

* Enhanced levels of gingival inflammation without increased levels of plaque accumulation occur in children at puberty.

* Estrogen receptors are found in basal and spinous layers of the epithelium and in fibroblasts and endothelial cells of small vessels in the connective tissue.

* Thus, gingiva appears to be a target organ for some of the steroid hormones.
The relationship between elevated levels of circulating sex hormones and prevalence of gingivitis in puberty is seen. Gingivitis peaks earlier in girls (11-13 years) than in boys (13-14 years).

It is characterized by pronounced inflammation bluish red discoloration, edema and enlargement.
II. ASSOCIATED WITH BLOOD DYSCRASIAS

LEUKEMIA-

* It is a malignant disease caused by the proliferation of the WBC forming tissues.
* It may be acute or chronic. WBC – granulocytes (myeloid), lymphocytes, or monocytes can get affected.
* Acute type leukemia- frequent in people under 20 years of age.
* Acute lymphoblastic leukemia- in children under 10 years.
* Factors that have been implicated to be of etiologic significance are radiation injury, chemical injury, genetic factors – Down’s syndrome, immune deficiency and viral infections.
Clinical features

* Gingiva appears as swollen, glazed, and spongy tissue which is red-deep purple in appearance with gingival bleeding.

* Diffuse enlargement of gingival mucosa seen.

* Moderately firm in consistency, but may be friable.

* Hemorrhage, either spontaneously or on slight irritation.

* Lethargy, malaise, sore throat, fever, skin infections that fail to heal, purpura, cervical lymphadenopathy, spleenomegaly, hepatomegaly and petechiae.
III. ASSOCIATED WITH NUTRITIONAL DEFICIENCY

SCORBUTIC GINGIVITIS

* Vitamin C deficiency causes hemorrhage, collagen degeneration and edema of the gingival connective tissue.

* Gingiva is bluish, soft, friable with smooth shiny surface.

* Hemorrhage occurring either spontaneously or slight provocation.

* Surface necrosis with pseudomembrane formation and necrosis occur as a result of infarcts created in the capillaries supplying the gingiva.
C. MODIFIED BY MEDICATION

DRUG INFLUENCED GINGIVAL ENLARGEMENT

* Overgrowth of gingiva is a well-recognized unwanted effect of a number of drugs.
* Most common are phenytoin, cyclosporine, nefidipine.
* Interdental papillae become nodular. Enlarged gingiva is pink, firm.
* Anteriorly most severely and frequently
* Patient’s physician- modify or change therapy.
NON PLAQUE INDUCED GINGIVAL DISEASES

A. VIRAL

ACUTE HERPETIC GINGIVOSTOMATITIS

* Organism is Herpes simplex virus (HSV) type 1
* Occurrence - Infants, children younger than 6 years of age.
* Clinical features - Diffuse erythematous, shiny involvement of the gingiva and the adjacent oral mucosa.
* Varying degrees of edema, gingival bleeding
* Discrete spherical gray vesicles which rupture and form painful small ulcers with red, elevated margin and depressed yellowish or grayish white central portion.
B. FUNGAL

CANDIDIASIS

- Overgrowth of *candida albicans*, usually after a course of antibiotics or as a result of congenital or acquired immunodeficiencies.

- Pathogenesis-The lesions of the oral disease appear as raised, furry, white patches, which can be removed easily to produce a bleeding underlying surface.
Clinical features:

* Pearly white or bluish white plaque present on oral mucosa which may extend to circumoral tissues.
* Painless and noticed on careful evaluation. They may be removed with little difficulty.
* Patient may complain of burning sensation.

Treatment:

* Infants and very young children- Nystatin 1ml (100,000U) dropped in to mouth for local action four times a day.
* Clotrimazole suspension (10mg/ml) 1 to 2 ml applied over affected areas four times daily.
* Systemic fluconazole suspension (10mg/ml) 6mg/kg body weight.
C. BACTERIAL

ACUTE NECROTIZING ULCERATIVE GINGIVITIS

- Developing countries, prevalence of ANUG is higher and disease frequently occurs in children.
- In India, 54-68% of the cases occurred in children below 10 years of age.

Clinical Characteristics:

- Punched out appearance due to ulcerated and necrotic papillae and gingival margins. Ulcers are covered by a yellowish-white or grayish slough termed psuedomembrane.
- Removal of the slough results in bleeding and underlying tissue becomes exposed.
Seldom associated with deep pocket formation as extensive gingival necrosis often coincides with loss of crestal alveolar bone.

Swelling of lymph nodes and increased bleeding tendency are often present.

Fever and malaise is not consistent.

Oral hygiene in these patients is usually poor.

Characteristic bacterial flora of spirochetes and fusobacteria has been isolated from the necrotic lesions.

Young age is one of the predisposing factors of ANUG.
**Treatment:**

- Perform debridement under local anesthesia.
- Remove pseudomembrane.
- Patient counselling should include specific oral hygiene instructions, instruction on proper nutrition,
- For any signs of systemic involvement, the recommended antibiotics are:
  - Amoxicillin, 375 mg TDS x daily for 7 days and/or
  - Metronidazole, 250 mg TDS x daily for 7 days
**GINGIVAL ABSCESS**

- Is a localized, painful rapidly expanding lesion that is usually of sudden onset

**Etiology** - Irritation from foreign substance.

**Clinical features**

- Localized, painful, rapidly expanding lesion. Limited to marginal gingiva or interdental papillae

- Early stage: red swelling with smooth shiny surface

- Within 24 hours to 48 hours - lesion will be fluctuant.

- Management: Incision and drainage
D. CONGENITAL ANOMALIES

CONGENITAL EPULIS-

* Congenital Epulis of newborn is rare gingival tumour that occurs along the alveolar ridge. Associated with abnormalities of teeth.
* Clinically- smooth well defined erythematous masses arising from gum pad. Size may be large enough to lift the upper lip.
* Unerupted teeth are not affected.

CONGENITAL GUM SYNECHIAE-

* It is characterized by congenital adhesions between different parts of oral cavity. It is rare type of disease. It causes difficulty in breathing and respiration soon after birth.
H. Gingival Manifestations Of Systemic Conditions

GINGIVAL LESIONS ASSOCIATED WITH CHICKEN POX

* Varicella herpes virus primarily affects individuals under the age of 15 years.
* In oral cavity small ulcers may develop in any area of the mouth, however, lesions are found most often on the palate, gingiva and buccal mucosa.
Periodontitis is inflammation of the gingiva and the adjacent attachment apparatus and is characterized by loss of connective tissue attachment and alveolar bone.
CHRONIC PERIODONTITIS

* Localized (<30% of teeth are involved)
* Generalized (>30% of teeth are involved)

CLINICAL FEATURES:
* Periodontal pockets
* Bone loss
* Bleeding
* Gingival inflammation
* Chronic periodontitis is more common in adults, while aggressive periodontitis may be more common in children and adolescents.
* It is characterized by a slow to moderate rate of progression that may include periods of rapid destruction.

* Furthermore, the severity of disease can be
  * Mild (one to two millimeters clinical attachment loss),
  * Moderate (three to four millimeters clinical attachment loss), or
  * Severe (≥ five millimeters clinical attachment loss).
AGGRESSIVE PERIODONTITIS

* The prevalence ranges from 0.84% to 26.9%.
* It may be generalized or localized affecting both primary and mixed dentition.
* Onset occurs between the eruption of the primary dentition and puberty.
* Page et al. (1983) first described “prepubertal periodontitis” as a disease entity and further subdivided it into a localized form of PP (LPP) and a generalized form of PP (GPP).
* Pathogenic bacteria that have been associated include Actinobacillus actinomycetemcomitans, Prevotella intermedia, Capnocytophaga species, Porphyromonas gingivalis and Eikenella corrodens.
LOCALIZED AGGRESSIVE PERIODONTITIS (LAgP)

- Interproximal attachment loss on at least two permanent first molars and incisors, with attachment loss on no more than two teeth other than first molars and incisors.
- May be self-limiting.
- Occurs in children and adolescents without clinical evidence of systemic disease and is characterized by the severe loss of alveolar bone around permanent teeth.
- Disease usually is localized to permanent first molars & incisors.
GENERALIZED AGGRESSIVE PERIODONTITIS (GAgP)

* Exhibits generalized interproximal attachment loss including at least three teeth that are not first molars and incisors.
* Disease of adolescents and young adults, often affects the entire dentition.
* Exhibit marked periodontal inflammation & have heavy accumulations of plaque and calculus.
* Subgingival sites harbour non-motile, facultatively anaerobic, Gram-negative rods including *Porphyromonas gingivalis*.
* The levels of *P. gingivalis* and *Treponema denticola* were significantly higher in GAgP.
NECROTIZING PERIODONTAL DISEASES (NPD)

* Significant findings - Presence of interproximal necrosis and ulceration and the rapid onset of gingival pain.
* Patients with NPD can often be febrile.
* Necrotizing ulcerative gingivitis/periodontitis sites harbor high levels of spirochetes and P. intermedia and invasion of the tissues by spirochetes occurs.
* Factors that predispose - viral infections (including HIV), malnutrition, emotional stress, lack of sleep etc.
Treatment involves mechanical debridement, oral hygiene instruction, and careful follow-up. Debridement with ultrasonics has been shown to be particularly effective and results in a rapid decrease in symptoms.

If the patient is febrile, antibiotics may be an important adjunct to therapy. Metronidazole and penicillin have been suggested as drugs of choice.
Periodontitis as a Manifestation of Systemic Diseases

PAPILLON-LEFEVRE SYNDROME (PALMOPLANTAR KERATODERMA WITH PERIODONTOSIS)

* Inherited as an autosomal recessive trait
* Mutation of the gene that produces the enzyme Cathespin C.
* Greater frequency in consanguineous offspring
CLINICAL FEATURES-

* Children are born looking completely normal.
* Teeth erupt in normal sequence, position, and time.
* At age 1, when primary teeth starting to erupt, the gum tissue is severely inflamed and generalized aggressive periodontitis accompany the teeth.
* By age 4, the child has lost all of their primary dentition.
* Gingival tissue in mouth goes back to healthy & normal.
* Eruption of the permanent dentition begins at normal age and in normal sequence.
* Patient will lose their permanent teeth and be completely edentulous by age 14-17.
Chediac Higashi Syndrome

- Rare, autosomal recessive disorder
- Abnormalities in the cytoplasmic granules fusion of phagosome and lysosome to form bactericidal phagolysosome is impaired. Primarily affects neutrophils.

CLINICAL FEATURES:
- Partial albinism, mild bleeding disorders, recurrent bacterial infections, rapidly destructive periodontitis
- Bone loss is usually generalized and severe.
- Patients do not respond to periodontal therapy, leading to premature loss of both deciduous and permanent dentitions.
Down syndrome

* Trisomy 21, mongolism, and autosomal chromosomal anomaly associated with impaired PMNs functions, connective tissue disorders, and gingival hyperinnervation.

Manifestations:

* (i) Gingivitis and periodontitis especially in lower anteriors.
* (ii) Enamel hypoplasia.
* (iii) Microdontia.
* (iv) Macroglossia.
* (v) Fissured tongue.
AAPD Guidelines for Periodontal Therapy

**Periodontal examination** - All patients should receive a comprehensive periodontal examination.

1. Extra- and intraoral examination to detect nonperiodontal oral diseases or conditions.

2. General periodontal examination to evaluate the topography of gingiva and related structures; to assess probing depth, recession, and attachment level; to evaluate the health of the subgingival area with measures such as bleeding on probing and suppuration.

3. Assessment of the presence, degree and/or distribution of plaque, calculus and gingival inflammation.
- Dental examination, including caries assessment, proximal contact relationships, the status of dental restorations and prosthetic appliances, and other tooth- or implant-related problems.
- Determination of the degree of mobility of teeth and dental implants.
- Occlusal examination.
- Interpretation of a satisfactory number of updated, diagnostic quality periapical and bite-wing radiographs or other diagnostic imaging needed for implant therapy.
- Evaluation of potential periodontal systemic interrelationships.
- Assessment of suitability to receive dental implants.
Treatment procedures

1. Chemotherapeutic agents. These agents may be used to reduce, eliminate, or change the quality of microbial pathogens; or alter the host response through local or systemic delivery of appropriate agent(s).

2. Resective procedures. These procedures are designed to reduce or eliminate periodontal pockets and create an acceptable gingival form that will facilitate effective oral hygiene and periodontal maintenance treatment. Soft tissue procedures include gingivectomy, gingivoplasty, and various mucogingival flap procedures. Osseous procedures include ostectomy and osteoplasty. Dental tissue procedures include root resection, tooth hemisection, and odontoplasty. Combined osseous and dental tissue procedures may be required for management of endodontic-periodontal lesions.
3. Periodontal regenerative procedures include: soft tissue grafts, bone replacement grafts, root biomodification, guided tissue regeneration, and combinations of these procedures for osseous, furcation, and recession defects. Periodontal reconstructive procedures include: guided bone regeneration, ridge augmentation, ridge preservation, implant site development, and sinus grafting.

4. Occlusal therapy, which may include: minor tooth movement, occlusal adjustment, splinting, or provision of devices to reduce occlusal trauma.
Children and adolescents are subject to several periodontal diseases. Although there is a much lower prevalence of destructive periodontal diseases in children than in adults, children can develop severe forms of periodontitis.

In young patients, the underlying cause for increased susceptibility and early onset of disease is unknown. These diseases are often familial, suggesting a genetic predisposition for aggressive disease.
REFERENCES

